6 Investing in a Healthier Future

A solid start for our children must be a national priority

Those who argue that societies cannot afford to make immediate investments in reducing environmental pollution fail to appreciate that there are some forms of harm that cannot be repaired. — Deborah Axelrod, Devra Lee Davis & Lovell A. Jones

As a nation, we value the wellbeing of our children. In addition to our natural urge to protect what we love, we know that at a societal level their success is key to a vibrant, secure future. Poll after poll shows more than 80 percent of Americans consider healthy children a top priority.[177] We must line up our practice and policies with these values.

Our current use of over a billion pounds of pesticides every year puts their wellbeing at risk and, as the science demonstrates, can derail brain and body development and rob them of their full potential.

If there were no other way to control pests, it would be one kind of choice: weighing one set of needed benefits against known and evolving harms. But given the fact that there are many proven ways to control pests without use of harmful chemicals, the choice is quite clear. It is time to have policies in place that better protect our children (see sidebar).

The National Research Council recommended swift action to protect children from pesticides nearly 20 years ago, and it has been 50 years since Rachel Carson sounded the initial alarm about the health harms pesticides can cause. What is standing in the way?

Pesticide industry well served by current policies

Our current system of industrial agriculture and pest control relies on chemical inputs sold by a handful of corporations. These multinational corporations wield tremendous control over the system, from setting research agendas[178] to financing, crop selection and inputs throughout the production and distribution chain.

Not surprisingly, these same corporations also hold significant sway in the policy arena, investing millions of dollars every year to influence voters, lawmakers and regulators at both the state and federal level to protect the market for pesticides.[179]

The result is agriculture, food and pest control systems that serve the interests of these corporations well. It does not, however, serve farmers, who have lost day-to-day control of their operations and are putting themselves and their families in harm's way. Farmworker interests are not served, as workers are continuously exposed to chemicals known to harm human health.

And the health of children across the country is compromised by exposure to pesticides used to control pests in agriculture and where they live, learn and play.

In short, the system is broken.

Prioritizing children's health requires real change

The best way to protect children from the harms of pesticides is to dramatically reduce the volume used nationwide. This would not only limit children's exposure during their most vulnerable years, it would also lower pesticide levels in the bodies of men and women of childbearing age—protecting current and future generations in one fell swoop. Those pesticides most harmful to children should be first on the list.

This is not a small change, and not a recommendation made lightly. Yet the science tells us the problem is serious and urgent, and that viable and safer alternatives are available. If we stay on our current path, our children will not reach their full potential as we continue to compromise their health.

U.S. Pesticide Rules

Overdue for overhaul?

A little over 100 years ago, Congress enacted our first national pesticide law. The 1910 Insecticide Act put labeling guidelines in place to protect farmers from "hucksters" selling ineffective, misbranded or adulterated pesticide products.

To this day, we control pesticides through a system of registration and labeling. The Federal Insecticide, Fungicide and Rodenticide Act (FIFRA), passed by Congress in 1947, is our primary national pesticide law. It has been updated several times in the last 65 years as the health and environmental effects of pesticides came into light, most significantly in 1972 and again in 1996.

It remains, however, a system of registration and labeling, and as such has significant shortcomings. Our current pesticide rules:

- Do not allow for quick response to emerging science;
- Do not assess risk based on real-world exposures;
- Rely heavily on corporate safety data that is not peer-reviewed; and
- Do not encourage the safest form of pest control.

In addition, enforcement of any guidelines or restrictions specified on product labels is relegated to state governments that rarely have adequate resources for the job. Overall, our current rules do not provide adequate tools to protect children from the harms of pesticide exposure.

Informed household food choices can help protect families and grow the market for food that is produced without harmful pesticides—encouraging more farmers to make this shift. And reducing household use of pesticides can provide immediate and long lasting benefits to children's health.* But the burden of protecting children from dangerous chemicals cannot rest solely with individual families. Policy change is required.

Recommendations: Effective policies urgently needed

To protect our children from the health harms of pesticides, policymakers must have much more effective tools. We believe such tools are most urgently needed as decisions are made about these three questions:

- Which pesticides are used in agriculture?
- Which pesticides are used in places children live, learn and play?
- How are farmers supported as they reduce reliance on pesticides?

We recommend the following policy changes in these three arenas:

1. Prevent the pesticide industry from selling agricultural products that can harm children's health

Given the wide-ranging susceptibility of children to pesticide exposures, plus the potential impacts on children from extremely low doses of toxic chemicals, the current approach to assessing and controlling risks of agricultural pesticides does not adequately protect our children.

Decisionmakers must have tools to remove an agricultural pesticide from the market quickly or deny a newly proposed pesticide market access when science suggests it can harm children's developing minds or bodies and there is evidence that children are likely to be exposed. Specifically, we recommend that rulemakers should:

- *Take swift action on existing pesticides:* If studies find a pesticide to be a neurodevelopmental or reproductive toxicant, endocrine disruptor or human carcinogen—*and* it has been measured in humans, in schools or homes, or as residues on food or in drinking water—EPA should target the pesticide for rapid phaseout, triggering USDA resources to assist rapid farmer transitions to safer pest control methods.†
- *Block harmful new pesticides:* EPA should not approve any new pesticide that scientific studies suggest is a neurodevelopmental or reproductive toxicant, endocrine disruptor or human carcinogen—including short-term "conditional" registrations.
- *Prevent harmful low-level exposures:* EPA should act on existing evidence that exposures to endocrine disrupting pesticides pose a particular danger to developing children; the long-delayed endocrine disruptor screening program (EDSP) should be swiftly implemented. At the current rate, it will be 2017 before the first set of *only 58 chemicals* are screened.

The best way to protect children from the harms of pesticides is to dramatically reduce the volume used nationwide.

The insecticide chlorpyrifos provides a clear example of the startling flaws in our regulatory system. Over 10 million pounds of the pesticide are still applied in agricultural

When Is There Enough Evidence to Act?

Scientific studies often identify a "link" or "association" between exposure to a particular pesticide and a specific health harm—but individual studies rarely demonstrate definitive causation. Epidemiological studies often lack statistical power, and case control and animal studies may miss key variables such as exposure timing.

A "weight of the evidence" approach recognizes that a body of scientific work will contain conflicting studies, but holds that when a number of well designed, robust studies come to similar conclusions, the findings should be considered valid.*

When such findings involve widespread, significant and irreversible health harms to our children, the bar for taking action should not be high. When credible evidence of harm emerges, a pesticide product should immediately be taken off the market until its manufacturer can prove its safety. Put simply, it is time the burden of proof shifted to the pesticide corporations, rather than regulators—and the public—as it currently stands.

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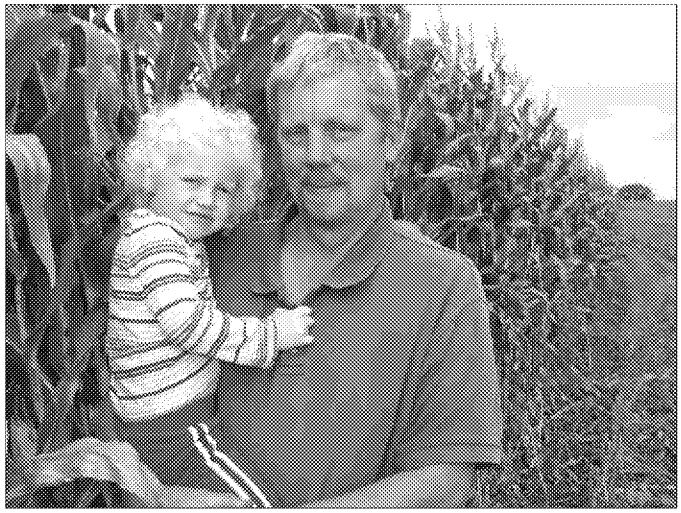
† See, for example, criteria and process for developing the "chemicals of high concern" list in Maine. http://www.maine.gov/dep/safechem/highconcern/chemicals.htm



Investing in farmers who grow food without relying on chemicals that harm children's health must be a national priority.

fields every year, more than a decade after household uses were withdrawn because of *clear dangers to children's developing brains.*[*] Yet children across the country continue to be exposed—in rural schools and communities, and by eating foods that have been treated with the neurotoxic chemical.

2. Protect children where they live, learn & play

Policymakers need strong tools to protect children from exposure to pesticides where they live, learn and play. Such protections will help keep developing bodies and minds healthy during the years they are most vulnerable to harm from chemical exposures.

We recommend rapid implementation of the following measures:

- *Kid-safe homes, daycares & schools:* EPA should withdraw approval of existing pesticide products and not approve new pesticides for use in homes, daycare centers or schools when scientific evidence indicates the chemicals are possible neurodevelopment or reproductive toxicants, endocrine disruptors or human carcinogens.
- *Safer parks & playgrounds:* State and local officials should enact policies requiring that all public playgrounds, playing fields and parks be managed without using pesticides that studies show are harmful to children's health.
- *Protective buffer zones:* State legislators should establish—or give local governments authority to establish—protective pesticide-free buffer zones around schools, daycare centers and residential neighborhoods in agricultural areas.
- *Healthier school lunches:* Local school districts, state agencies and USDA's Farm-to-school program should provide schools with incentives to procure fresh, local fruits and vegetables that have been grown without pesticides that studies show are harmful to children's health.

3. Invest in farmers stepping off the pesticide treadmill

Investing in farmers who grow food without relying on chemicals that harm children's health must be a national priority. Specifically:

- *Corral resources for farmers:* Federal and state officials should mobilize and coordinate existing resources to help farmers adopt well-known, effective pest management strategies that reduce reliance on pesticides. USDA, EPA and many state agencies and universities have important programs—research, outreach and education—with this stated aim that could be ramped up in complementary ways.
- *Increase investment in innovative farming:* Congress should authorize significant funding for programs supporting farmers' adoption of sustainable practices that reduce use of harmful pesticides. Existing programs receive a small fraction of the funding supplied to programs serving conventional growers.
- *Set use reduction goals:* EPA and USDA should set specific and aggressive national pesticide use reduction goals, focusing first on pesticides studies show to be harmful to children.[†] To track progress toward this goal, farmers should work with applicators and pest control advisors to report their pesticide use to a nationally searchable database.[‡]
- *Source for children's health:* Food distributors should require that their suppliers limit use of pesticides that harm children's health.

Effective agroecological methods exist for production of all major crops—but these approaches are often knowledge-intensive, requiring significant training as well as real changes in farm operation.[§] Growers need direct support to make the shift away from pesticide reliance, including provision of hands-on field training and technical advice from independent experts as well as incentives to invest in agroecological practices.

These proposals are all commonsense measures in the face of clear evidence that our children's wellbeing is at risk. It's time to muster the political will and prioritize the health of our children, grandchildren and future generations.

* Chlorpyrifos was phased out for household use after studies clearly indicated that exposed children had smaller head circumference, a known indicator of reduced cognitive function.

† See Appendix B.

‡ Pesticide use reporting is already in place in California; lessons learned from implementation of this program (established in 1990) should inform and enable rapid adoption of a federal use reporting system.

§ Agroecological practices are based on the application of intricate place-based knowledge of soil/plant/animal interactions designed to prevent or minimize pest problems. Farmers are successfully using such practices in virtually every crop now grown in the U.S.

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Appendix A

More Science: Key study descriptions

Our intention in undertaking this review was not to conduct a comprehensive evaluation of the evidence. The body of scientific literature exploring how pesticides affect children's health is wide, deep and decades long.

Our goal is to provide a snapshot of recent findings, coming fast and furious in the just the past few years, that—taken together—provide compelling reason for concern about the impact of pesticides on our children's health.

In the report itself we highlight a few of the key findings for each health effect, focusing on studies that were particularly compelling, and/or represented other studies we reviewed with similar findings. We simplified descriptions of each study to provide a basic sense of how the research was conducted and what researchers found. Here in Appendix A we provide a bit more detail on some of the key studies described above, as well as additional studies. Study descriptions are organized by health effect, and alphabetically by author within each category.

Brain & nervous system harms (reduced cognitive function, autism, ADHD)

Bouchard M.F., D.C. Bellinger, R.O. Wright and M.G. Weisskopf."Attention-deficit/ hyperactivity disorder and urinary metabolites of organophosphate pesticides." ***Pediatrics*** **2010. 125(6): e1270–e1277.**

This study examines the association between urinary concentrations of organophosphate metabolites and ADHD in children eight to 15 years of age. Researchers analyzed cross-sectional data from the National Health and Nutrition Examination Survey for 1139 children representative of the U.S. population. Urinary DMAP metabolite levels (which are an indicator of exposure to OP pesticides), an ADHD assessment, and household surveys were used in the analysis. The data support the hypothesis that organophosphate exposure, at levels common among U.S. children, may contribute to ADHD prevalence.

Eskenazi B., K. Huen, A. Marks, K.G.Harley, A. Bradman, D.B. Barr, et al. "PON1 and Neurodevelopment in Children from the CHAMACOS Study Exposed to Organophosphate Pesticides in Utero." ***Environ Health Perspect.*** **Aug 2010 118: 1775-1781. See http://dx.doi.org/10.1289/ehp.1002234.**

The enzyme paraoxonase 1 (PON1) detoxifies metabolites of some organophosphate (OP) pesticides, andPON1 genetic polymorphisms influence enzyme activity and quantity. The study authors investigated whether PON1 genotypes and enzyme activity levels in mothers and their children were linked to neurodevelopmental changes, and whether PON1 levels and genotypes had an effect on the association of *in utero* exposure to OP pesticides (as assessed by maternal urinary concentrations of dialkyl phosphate metabolites, a marker of OP pesticide exposure) and neurodevelopment and behavior. The researchers found that of the 353 two-year-olds assessed, children with a certain variation of PON1 (the $PON1_{-108T}$ allele) scored more poorly on the Mental Development Index and somewhat lower on the Psychomotor Development Index. The authors concluded that while the variations of PON1 were associated with outcomes in child neurobehavioral development, additional research is needed to confirm whether it modifies the relation with *in utero* exposure to OP pesticides.

Pessah I.N., P.J. Lein. "Evidence for environmental susceptibility in autism" in: ***Autism,*** **(Zimmerman AW, ed). Totowa, NJ: Humana Press 2008 409–428.**

The authors aim to illustrate how research into the pathophysiology and genetics of autism may inform the identification of environmental susceptibility factors that promote adverse outcomes in brain development. They highlight three examples of gene-environment interactions that are likely to contribute to autism risk, including: (1) pesticides that interfere with the neurotransmitter acetylcholine; (2) pesticides that interfere with γ-aminobutyric acid (GABA) neurotransmission; and (3) persistent organic pollutants that directly

A Study by Any Other Name...

Epidemiological study: A study of distribution or patterns in health trends or characteristics and their causes or influences in specific populations. Includes both case-control and all types of cohort studies.

Case-control study: Compares a "case" group (e.g., U.S. children ages 0–14 with cancer) with a group serving as a control (e.g., cancer-free U.S. children ages 0–14).

Cohort study: Profiles a specific population where shared exposure may be assumed, such as occupational exposure to pesticides among farmworkers.

Prospective cohort study: Follows a group that is slightly different in some respects. (i.e., studying a cohort of pesticide applicators who use varying protective methods while working with pesticides.)

Longitudinal cohort study: Tracks a specific group over time. For example, a UC Berkeley study on the central California coast has followed a specific group of children from conception through adolescence.

Meta-analysis: Pulls together several studies on the same topic and does further statistical analysis on the basic findings.

Review: Examines the "state of the science" and often provides evaluation of conflicting pieces of data. Review authors give their view on what is currently happening in the field.

alter calcium ion (Ca^{2+}) signaling pathways and Ca^{2+}-dependent effectors. If both genetic factors and environmental ones converge to interrupt the same neurotransmitter or signaling systems at critical times during development, adverse effects can be amplified.

Rauh V.A., F.P. Perera, M.K. Horton, R.M. Whyatt, R. Bansal, X. Hao X, et al. "Brain anomalies in children exposed prenatally to a common organophosphate pesticide." ***Proc Natl Acad Sci*** **2012 109(20):7871-6.**

This study investigated associations between prenatal exposure to chlorpyrifos and brain morphology (examining brain structure). With a sample of 40 children—who experienced low prenatal exposure to tobacco smoke and polycyclic aromatic hydrocarbons—20 subjects with high chlorpyrifos exposure were compared to 20 low-exposure subjects. The data revealed a significant association between prenatal exposure to chlorpyrifos, at standard use levels, and structural changes in the developing human brain. High exposure was associated with the enlargement of several areas of the brain and in preliminary analyses, the reversal of sex differences or a lack of expected sex differences.

Shafer, T.J., D.A. Meyer and K.M. Crofton. "Developmental Neurotoxicity of Pyrethroid Insecticides: Critical Review and Future Research Needs." ***Environmental Health Perspectives*** **113, no. 2 Oct 2004: 123–136 .**

A review of pyrethroid insecticides and the data related to potential developmental neurotoxic effects of pyrethroids, with recommendations for improving study design and statistical analyses. The review discusses the various effects on voltage-sensitive sodium channels, which are a primary target of pyrethroids.

Childhood cancers

Carozza S.E., B. Li, K. Elgethun and R. Whitworth."Risk of childhood cancers associated with residence in agriculturally intense areas in the United States." ***Environ Health Persp*** **2008 116(4): 559–565.**

Researchers from the U.S. evaluated whether children under the age of 15 who live in a county associated with greater agriculture production—and hence, exposure to pesticide drift—experienced different risk rates for developing cancer. Using incidence data for U.S. children provided by the North American Association of Central Cancer Registries, researchers were able to compare county-level, sex- and age-specific rates of childhood cancer with agricultural census data containing county acreage, percent cropland, and percent acres for specific crops. The data revealed statistically significant increase in risk for many types of childhood cancers for residents living in those counties with a moderate to high level of agricultural activity. Risk for different cancers varied by type of crop; for example, there was increased risk of non-Hodgkin lymphoma and thyroid cancer associated with residence at diagnosis in counties that produced corn or oats.

Infante-Rivard C, S. Weichenthal. Pesticides and childhood cancer: an update of Zahm and Ward's 1998 review. ***J Toxicol Environ Health B Crit Rev*** **2007 10(1): 81–99.**

Infante-Rivard and Weichenthal reviewed the epidemiological and ecological studies published since the 1998 Zahm and Ward review. The authors found that15 case-control studies, four cohort studies, and two ecological studies have been published since this review, and 15 of these 21 studies reported a statistically significant increase in risk of childhood cancer among children whose parents were experienced occupational pesticide exposure. These studies found that the risk of all childhood cancers increased with the frequency of maternal exposure to herbicides and plant insecticides. Furthermore, maternal and paternal exposure to insecticides and herbicides up to five years before having a child increased risk of all childhood brain tumors, astroglial tumors, non-Hodgkin's lymphoma, primitive neuroectodermal tumors, and other glial tumors. Parental occupation in agriculture is also associated with an increased risk of Ewing's sarcoma. The authors conclude that evidence supports an association between at least some pesticide exposure and childhood cancer.

Kristensen, P., A. Andersen, L.M. Irgens, A.S. Bye and L. Sundheim. "Cancer in Offspring of Parents Engaged in Agricultural Activities in Norway: Incidence and Risk Factors in the Farm Environment." ***International Journal of Cancer. Journal International Du Cancer.*** **Jan 1996 65 (1): 39–50.**

A cohort study in Norway of 323,359 children born between 1952–1991 reported that children 0-14 years had a nearly doubled risk for brain tumors and a more than tripled risk for neuroepithelial tumors except for astrocytomas associated with pesticide purchase. These associations were stronger when sub-groups, such as growing up on the farm, were considered. Offspring born April–June showed a clustering of neuroepithelial brain tumors, suggesting that paternal exposure during periods of increased pesticide application, from 0–3 months before conception, may have been a factor.

Meinert, R., J. Schuz, U. Kaletsch and J. Michaelis. "Leukemia and Non-Hodgkins Lymphona in Childhood and Exposure to Pesticides: Results of a Register-based Case-Control Study in Germany." ***Am Journal of Epidemiology*** **2000. 151 (7): 639-646.**

A case-control study conducted in Germany from 1993-1997 found parental occupational exposure to be related to childhood cancer regardless of period of exposure and type of cancer, which the authors point out might be due to different recall of past exposures between parents of cases and parents of controls. Residential insecticide use was associated with childhood lymphoma, both professional exterminator and parental usage were significantly associated with increased risk.

Nielsen S.S., R. McKean-Cowdin, F.M. Farin, E.A. Holly, S. Preston-Martin and B.A. Mueller. "Childhood brain tumors, residential insecticide exposure, and pesticide metabolism genes." ***Environ Health Persp*** **2009 118(1): 144-149.**

Researchers in California and Washington found evidence of increased risk of childhood brain tumors (CBT) associated with certain genetic polymorphisms when kids were exposed to insecticides. Strong interactions between genotype and insecticide exposure during childhood was observed. Among exposed children, CBT risk increased per $PON1_{-108T}$ allele, whereas among children never exposed, CBT was not increased. Nielsen et al. concluded childhood exposure to organophosphorus pesticides coupled with a reduced ability to detoxify these pesticides, may be associated with CBT.

van Wijngaarden E, P.A. Stewart, A.F. Olshan, D.A. Savitz and G.R. Bunin. "Parental occupational exposure to pesticides and childhood brain cancer." *Am J Epidemiol* 2003. 157(11): 989–997.

Researchers from the U.S. evaluated parental exposure to pesticides at home or on the job in relation to the occurrence of brain cancer in children. The sample consisted of children diagnosed with cancer and matching controls from four U.S. states. Interviews were performed with the biological mothers of the subjects to assess the residential and occupational exposure to pesticides in the two years before the child was born. The data revealed a significant risk of astrocytoma associated with residential use and exposure to herbicides. Combining parental exposures to herbicides form both residential and occupational sources, the elevated risk remained significant.

Birth defects

Brender, J.D., M. Felkner, L. Suarez, M.A. Canfield and J.P. Henry. "Maternal Pesticide Exposure and Neural Tube Defects in Mexican Americans." *Annals of Epidemiology*. 2010 20(1): 16–22.

Researchers investigated the relationship between maternal pesticide exposures and neural tube defects (NTDs) in offspring comparing to groups of Mexican American women (184 in case group, 225 for comparison). After adjusting for differences in maternal education levels, smoking, and folate intake during pregnancy, women who reported using pesticides in their homes or yards were twice as likely to have children with NTDs than women not reporting exposures (95% confidence interval [CI], 1.2–3.1) Case-women were also more likely to live within ¼ mile of agricultural fields. As possible sources of pesticide exposure increased, risk of NTDs also increased. Associations were stronger for risk of anencephaly than for spina bifida.

Garry V.F., M.E. Harkins, L.L. Erickson, L.K. Long-Simpson, S.E. Holland and B.L. Burroughs. "Birth defects, season of conception, and sex of children born to pesticide applicators living in the Red River Valley of Minnesota, USA." *Environ Health Persp* 2002. 110(3): 441–449.

A cross-sectional study performed in the Red River Valley of Minnesota examined the reproductive health outcomes in 695 farm families (analyzed data from 1,532 children) from parent-reported birth defects. Researchers determined conceptions in the spring time led to significantly more children born with birth defects, compared to children conceived in any other season. Their data suggests environmental agents present in the spring, like herbicides, have an adverse effect on the birth defect rate. Furthermore, the data revealed an association between fungicide exposure and the determination of child sex—affecting the survival rate of the male fetus (female to male birth ration is 1.25 to 1).

Gaspari L., F. Paris, C. Jandel, N. Kalfa, M. Orsini, J.P. Daures and C. Sultan. "Prenatal environmental risk factors for genital malformations in a population of 1442 french male newborns: a nested case-control study." *Hum Reprod* 2011. 26(11): 3155–3162.

Researchers from France analyzed a physician's examinations and parental interviews for 1442 full-term newborn males in southern France to identify risk factors for male external genital malformations, with a focus on parental occupational exposure to endocrine disrupting chemicals, such as organochlorine pesticides. Infants were examined for cryptochidism, hypospadias, and micropenis, while a questionnaire asked parents about the pregnancy, personal characteristics, lifestyle, and occupational exposure to EDCs. In total, 39 cases of genital malformation were reported (2.70%). A significant relationship was observed between newborn cryptochidism, hypospadias or micropenis and parental occupational exposure to pesticides with the odds of genital malformation increasing 4.41-fold. These data supports the hypothesis that prenatal contamination by pesticides may be a potential risk factor for newborn male external genital malformation.

Rocheleau, C.M, P.A. Romitti and L.K. Dennis. "Pesticides and Hypospadias: a Meta-analysis." *Journal of Pediatric Urology*. Feb 2009 5(1): 17–24.

A meta-analysis of studies done in 7 different countries (Canada, Denmark, Italy, Netherlands, Norway, Spain, US) indicated a 36% increased risk of hypospadia with maternal occupational exposure and a 19% increased risk of hypospadias with paternal occupational exposure.

Winchester PD, Huskins J, Ying J. 2009. Agrichemicals in surface water and birth defects in the United States. *Acta Paediatr* 98(4): 664–669.

Researchers from Indiana and Ohio compared water data from the USGS National Water Quality Assessment (NAWQA)—measuring the levels of nitrates, atrazine, and other pesticides in surface water—and Centers for Disease Control data detailing monthly pregnancy and birth outcome outcomes. The data reveal that between 1996 and 2002 women in the US were significantly more likely to give birth to a child with birth defects if conception had occurred in the months of April through July. NAWQA surface water samples indicate that concentrations of atrazine, nitrates, and other pesticides were also higher in the months of April through July. This correlation was statistically significant, demonstrating elevated concentrations of agrichemicals in surface water coincided with a higher risk of birth defects among live births for children conceived between April and July.

Early puberty

Aksglaede L., K. Sorensen, J.H. Petersen, N.E. Skakkebaek and A. Juul. "Recent decline in age at breast development: the Copenhagen puberty study." *Pediatrics* 2009. 123(5): e932-939.

Researchers from Denmark collected data from 2095 females aged 5.6 to 20 years in two Copenhagen cohorts (1991–1993 and 2006–2008) to examine differences in breast development. Using the most accurate method of palpation, Aksglaede et al. found the onset of puberty—defined as the mean estimated age at the attainment of glandular breast tissue—occurred significantly earlier in the 2006 cohort. The ages at which menarche and pubic hair development occurred also slightly decreased in the 2006 cohort. As a result of these timing changes in early and later markers of puberty, the length of puberty appears to have increased. The authors interpreted these observations as indicative of gonadotropin-independent estrogenic actions at the level of breast development, rather than an earlier activation of the pituitary-gonadal axis. These changes in timing could not be explained by alterations in reproductive hormones and BMI, suggesting other factors involved need to be explored.

Gladen B., N. Ragan and W. Rogan. "Pubertal growth and development and prenatal and lactational exposure to polychlorinated biphenyls and Dichlorodiphenyl Dichloroethene." *Pediatrics* 2000. 136(4): 490-496.

Researchers from the National Institute of Environmental Health Sciences explored the relationship between prenatal and early-life exposure to PCBs and DDE on children. This is one of a very few studies examining environmental contaminants and male puberty onset. Using 594 children from the North Carolina Infant Feeding Study cohort, they found no effect on the ages at which puberty began. However, the height and weight (adjusted for height) of boys at puberty increased with transplacental exposure to DDE.

Massart F., P. Seppia, D. Pardi, S. Lucchesi, C. Meossi, L. Gagliardi et al. "High incidence of central precocious puberty in a bounded geographic area of northwest Tuscany: an estrogen disrupter epidemic?" *Gynecol Endocrinol* 2005. 20(2): 92–98.

Researchers in Italy preformed an analysis of central precocious puberty (CPP) distribution in northwest Tuscany (NWT). The overall incidence rate of sexual precocity is estimated at 10–20 per 100, a rate similar to that found in four of the cities in the NWT sample; however 47 percent of the CPP cases found in NWT were in the Viareggio area, a rate of 161 per 100,000. This area hosts a high density of navy yards and greenhouses—consequently it is at higher risk of chemical estrogen pollution. As this population represented only 13.73 percent of the total population of NWT, living in this area significantly increased the risk of CPP. The definite geographic distribution of CPP in this suggests that environmental involvement/pollution may be a major determinant of CPP development.

Nebesio T and O. Hirsh Pescovitz. " Historical perspectives." *Endocrinologist* 2005. 15(1):44-48.

Nebesio and Pescovitz reviewed reports alleging endocrine disruptors blamed for altering the age of normal puberty, including an examination of studies implicating pesticides and accidental environmental exposures. Studies reviewed include two seminal studies on early puberty in girls: Vasiliu et al.'s (2004) examination of the Michigan anglers cohort daughters and Krzstevska-Konstantinova et al.'s (2001) examination of precocious puberty in native and non-native Belgian girls. Nebesio and Hirsch Pescovitz (2005) also review Boneh et al. (1989), who examined cases of girls with precocious sexual development from Jerusalem over a 10-year time period and found strong evidence for a seasonal increase in incidences of early sex development observed (from April–June). Seasonal pesticide usage was a potential cause, but the reasons for this were unknown.

Steingraber S. 2007. *The falling age of puberty in U.S. girls: what we know, what we need to know.* The Breast Cancer Fund.

In this report Steingraber suggests that pubertal onset and menarche are two sexual maturation processes that appear to be becoming uncoupled, therefore increasing the length of puberty in girls. The author cites environmental contaminants as the cause in light of recent evidence suggesting even minimal exposure to an endocrine disruptor on sex hormones can have a profound consequence in childhood.

Obesity & diabetes

Baillie-Hamilton, P.F. "Chemical toxins: a hypothesis to explain the global obesity epidemic." *J Altern Complement Med* 2002 8(2): 185–192.

Hamilton puts forth a new hypothesis to explain the global obesity epidemic: chemical toxins. Overeating and inactivity do not fully explain the current trend in obesity. Baillie-Hamilton calls for an examination of environmental causes rather than genetic factors. The sympathetic nervous system is perhaps the key weight-controlling system, and is targeted by many of the commonest synthetic chemicals. Numerous widely used synthetic chemicals induce weight gain, including pesticides (specifically organochlorines and organophosphates). They do so by disrupting major weight controlling hormones, altering levels and sensitivity to neurotransmitters, interfering with metabolic processes, and causing widespread damage to body tissues. These interferences change appetite, food efficiency, and the metabolism of fats, proteins, and carbohydrates.

Janesick, A. and B. Blumberg. "Endocrine Disrupting Chemicals and the Developmental Programming of Adipogenesis and Obesity." *Birth Defects Research Part C: Embryo Today*: Reviews 2011. 93, no. 1: 34–50.

This review article explores possible explanations for the variation in individual propensity to gain weight and accrue body mass, even at identical levels of caloric input. The authors review evidence from clinical, epidemiological, and biological studies showing that obesity is largely programmed early in life, including prenatally. They examine the environmental obesogen hypothesis, which holds that "prenatal or early life exposure to certain endocrine disrupting chemicals can predispose exposed individuals to increased fat mass and obesity. Obesogen exposure can alter the epigenome of multipotent stromal stem cells, biasing them toward the adipocyte lineage at the expense of bone." Individuals exposed to obesogens early in life or prenatally might thus experience changes in their stem cell compartment, which in turn influences adipogenic fate

Lee D.H., I.K. Lee, K. Song, M. Steffes, W. Toscano, B.A. Baker and D.R. Jacobs."A strong dose-response relation between serum concentrations of persistent organic pollutants and diabetes: results from the National Health and Examination Survey 1999-2002." *Diabetes Care* 2006 29(7): 1638–1644.

Researchers performed a cross-sectional examination of the association between serum concentrations of six POPs (selected because they were detectable in greater than 80 percent of participants) and diabetes prevalence. After adjustments were made for confounding variables (age, sex, race and ethnicity, poverty income ratio, BMI and waist circumference) diabetes prevalence was strongly positively associated with lipid adjustment serum concentrations of all six POPs tested for in the sample of 2,016 adult participants from the National Health and Nutrition Examination Survey 1999–2002. Furthermore, the association between POPs and diabetes was much stronger among obese subjects compared to lean subjects.

Lee, D.H., M.W. Steffes, A. Sjödin, R.S. Jones, L.L. Needham, D.R. Jacobs. "Low dose organochlorine pesticides and polychlorinated biphenyls predict obesity,

dyslipidemia, and insulin resistance among people free of diabetes." *PLoS One* 2011 6(1): e15977.

In a follow up study to their 2010 study of low-dose persistent organic pollutant (POP) exposure and prediction of type 2 diabetes, Lee et al. conducted a nested case-control study to explore the relationship between serum concentrations of POPs and adiposity, dyslipidemia, and insulin resistance among people confirmed to be diabetes free (assessing study subjects on 5 occasions over 20 years). Researchers concluded that simultaneous exposure to various OC pesticides and PCBs in the general population may contribute to the development of obesity, dyslipidemia, and insulin resistance—common precursors of type 2 diabetes and cardiovascular diseases—among those without diabetes. POPs exposure may also contribute to excess adiposity and other dysmetabolic conditions. Ten POPs were found to predict future higher triglycerides and 14 POPs predicted lower HDL-cholesterol. Among organochorine pesticides, p,p'-DDE most consistently predicted higher BMI, triglycerides and HOMA-IR, as well as a lower HDL-cholesterol at year 20.

Newbold R.R., E. Padilla-Banks, R.J. Snyder, T.M. Phillips and W.M. Jefferson. "Developmental exposure to endocrine disruptors and the obesity epidemic." Reprod Toxicol 2007. 23(3): 290–296.

Research from the US has shown an association between exposure to environmental endocrine disrupting chemicals with the development of obesity. Researchers utilize an animal model of developmental exposure to diethylstilbestrol (DES)—a potent perinatal endocrine disruptor with estrogenic activity—to study the mechanisms involved in programming an organism for obesity. Their data supports the idea that brief exposure early in life to environmental endocrine disrupting chemicals, especially those with estrogenic activity, like DES. These chemicals may contribute to overweight and obesity as well as other obesity-associated diseases (type 2 diabetes and cardiovascular disease). This research complicates the current understanding of obesity and necessitates a consideration of more complex factors, including environmental chemicals.

Asthma

Hernández A.F., T. Parrón and R. Alarcón. "Pesticides and asthma." *Curr Opin Allergy Clin Immunol* 2011 11(2): 90–96.

Hernández et al. performed a review of clinical and epidemiological studies that link exposure to pesticides, asthma attacks, and an increased risk of developing asthma. These authors concluded that while many pesticides are sensitizers or irritants, their potential to sensitize is limited. However, more importantly, pesticides may increase the risk of developing asthma, exacerbate a previous asthmatic condition or even trigger asthma attacks by increasing bronchial hyper-responsiveness.

Salam MT, Y.F. Li, B. Langholz, F.D. Gilliland."Early-life environmental risk factors for asthma: findings from the Children's Health Study." *Environ Health Perspect* 2003 112(6): 760–765.

Researchers from the University of Southern California selected 4,244 subjects from the Children's Health Study conducted in 12 southern California communities to measure the relationship between childhood environmental exposures and asthma risk. Matching those subjects diagnosed with asthma before age five with asthma-free counterparts that acted as controls (matched for age, sex, community of residence, and in utero exposure to maternal smoking), the authors concluded that environmental exposures during the first year of life are associated with an increase in the risk for early-onset persistent asthma, a subtype of asthma associated with long-term morbidity. Compared to never-exposed children, children exposed to herbicides within the first year of life had a 4.6-fold increased risk of asthma and children exposed to pesticides had a 2.4-fold increase in risk—considered together children exposed to any pesticide or herbicide in the first year of life experience a 2.53-fold higher risk of asthma compared to children who were never exposed to either of those.

Salameh P.R., I. Baldim, P. Brochard, C. Raherison, B.A. Saleh and R. Salamon. "Respiratory symptoms in children and exposure to pesticides." *Eur Respir J* 2003 22(3): 507–512.

Public health researchers from Lebanese University in Lebanon and Victor Segalen Bordeaux II University in France conducted a cross-sectional study to evaluate if exposure to pesticides resulted in chronic effects on the respiratory health of Lebanese children. From 19 public schools, 3,291 randomly selected school children—aged five to 16 years—revealed exposure (residential, paraoccupational, and domestic) to pesticides was significantly associated with respiratory disease (1.82-fold higher) and chronic respiratory symptoms such as chronic phlegm, chronic wheezing, and wheezing at any point (the only exception was chronic cough). Twelve percent of the sample reported a chronic respiratory disease and of those, 84 reported a medically confirmed asthma diagnosis (2.6 percent of the sample).

Sunyer J, M. Torrent, R. Garcia-Esteban, N. Ribas-Fitó, D. Carrizo, I. Romieu et al. "Early exposure to Dichlorodiphenyldichloroethylene, breastfeeding and asthma at age six." *Clin Exp Allergy* 2006 36(10): 1236–1241.

Researchers from Spain and the United Kingdom conducted a longitudinal study from a sample of 468 Minorcan children (Balearic Island in the northwest Mediterranean sea with no local pollution sources) to examine the association between prenatal exposure to DDE and other organochlorine compounds and asthma. Asthma was defined as the presence of a wheeze, persistent wheezing, or parental report of doctor-diagnosed asthma at age four. All children were born with quantifiable levels of DDE and PCB compounds. Wheezing at age four was reported for 11.6 percent of all children. Wheezing at four years of age increased with DDE concentration, particularly at the highest quartile, which was also found for persistent wheezing. This association was maintained even after adjusting for potential confounding variables. These results corroborated the association established between DDE and asthma in German school children conducted by Karmaus et al. in 2001.

Appendix B: Top Pesticides Used in Agriculture & at Home

Key
? -- Insufficient data
ND -- No data available
I -- Insecticide
H -- Herbicide
F -- Fungicide
PGR -- Plant growth regulator
FUM -- Fumigant

Table B-1: Most Commonly Used Pesticide Active Ingredients - Agriculture Listed by volume of use[1]

Pesticide & use level range (millions of lbs active ingredient)	PAN HHP[2]	Type	High[3] acute toxicity	Carcin-ogen	Acute neuro-toxicant (ChE inhibitor)	Devel. or reprod. toxicant	Endocrine disruptor	Primary crops	Food residues[4]
Glyphosate (180-185)		H				?	?	Hay/pasture, soybeans, corn	ND
Atrazine (73-78)	Y	H		Y		?	suspected	Corn, sugarcane	Spinach, wheat, onions, lettuce, water
Metam-sodium (50-55)	Y	FUM	Y	Y		Y	suspected	Potatoes, carrots, tomatoes, onions, peanuts	ND
Metolachlor, (S) (30-35)	Y	H		possible		?	suspected	Tomatoes, beans, corn, cotton	Oats, celery, water, corn
Acetochlor (28-33)	Y	H		Y		?	suspected	Corn, popcorn	Water
Dichlorpropene (27-32)		FUM	Y	Y		?	?	Strawberries, sweet potatoes, tree nuts	
2,4-D (25-29)	Y	H		possible		?	suspected	Grasses, wheat, citrus fruits, tree nuts	Potatoes, water
Methyl bromide (11-15)	Y	FUM	Y			Y	suspected	Tomatoes, strawberries, almonds, peppers, watermelon, cucumbers	ND
Chloropicrin (9-11)	Y	FUM	Y	?		?	?	Tobacco, tomatoes, strawberries, bell peppers	ND
Pendimethalin (7-9)	Y	H		possible		?	suspected	Soybeans, corn, cotton, peanuts	Carrots, collard greens, kale
Ethephon (7-9)		PGR			Y	?	?	Cotton, walnuts, grapes, tomatoes	ND
Chlorothalonil (7-9)	Y	F	Y	Y		?	?	Tomatoes, watermelons, onions	Cranberries, celery, green beans
Metam Potassium (7-9)		FUM	Y	Y		Y	?	Lettuce, potatoes	ND
Chlorpyrifos (7-9)	Y	I			Y	?	suspected	Tree nuts, apples, alfalfa, broccoli, citrus, grapes, sweet corn	Apples, bell peppers, cranberries, kale, grapes, peaches
Copper Hydroxide (6-8)		F				?	?	Tree nuts, grapes, peaches	ND
Simazine (5-7)	Y	H				Y	suspected	Corn, citrus, grapes, tree nuts	Blueberries, kale, water, oranges
Trifluralin (5-7)	Y	H		possible		?	suspected	Soybeans, cotton, green beans, broccoli, tomatoes	Carrots, spinach, wheat, soybeans, broccoli
Propanil (4-6)	Y	H		possible		?	suspected	Rice, oats, barley, wheat	Wheat
Mancozeb (4-6)	Y	F		Y		Y	suspected	Apples, tomatoes, onions, watermelon	ND
Acephate (2-4)	Y	I		possible	Y	?	suspected	Cotton, tobacco, cranberries, mint	Green beans, bell peppers
Diuron[5] (2-4)	Y	H		Y		Y	suspected	Oranges	Asparagus, oranges, water, potatoes
MCPA (2-4)	Y	H	Y	possible		?	?	Flax, barley, wheat, rice	water
Paraquat (2-4)	Y	H	Y			?	suspected	Corn, soybeans, cotton, apples	ND
Dimethenamid (2-4)	Y	H		possible		?	?	Corn, soybeans, sugarbeets	Soybeans, water

Table B-2: Most Commonly Used Pesticide Active Ingredients – Home & Garden
Listed by volume of use

Pesticide & use level range (millions of lbs active ingredient)	PAN HHP	Type	High acute toxicity	Carcinogen	Acute neurotoxicant (ChE inhibitor)	Devel. or reprod. toxicant	Endocrine disruptor
2,4-D (8-11)	Y	H		possible		?	suspected
Glyphosate (5-8)		H				?	?
Carbaryl (4-6)	Y	I		Y	Y	Y	suspected
Mecoprop-P (MCPP) (4-6)	Y	H		possible		?	?
Pendimethalin (3-5)	Y	H		possible		?	suspected
Pyrethroids[6] (2-4)	Y	I	Y	Y		Y	suspected
Malathion (2-4)	Y	I	Y	possible	Y	Y	suspected
Dicamba (1-3)		H				Y	?
Malathion (2-4)	Y	I	Y	possible	Y	Y	suspected
Trifluralin (1-3)	Y	H		possible		?	suspected
Pelargonic Acid (< 1)		H/F		?		?	?

Notes

1 See Table 3.6 and 3.7 in *Pesticide Industry Sales & Usage, 2006 and 2007 Market Estimates*, U.S. EPA, Washington, DC Feb 2011. See www.epa.gov/opp00001/pestsales/07pestsales/market_estimates2007.pdf. Aldicarb was removed from the list as registration was withdrawn in 2010.

2 PAN International has compiled and published a list of Highly Hazardous Pesticides (HHPs) that are harmful to human health and the environment, and targeted for global reduction and elimination. See www.panna.org/issues/publication/pan-international-list-highly-hazardous-pesticides.

3 PAN's online pesticide database provides an explanation of these categories and additional toxicity, use and regulatory information for these and other pesticides. See www.pesticideinfo.org.

4 Based on USDA's Pesticide Data Program, as listed on www.whatsonmyfood.org.

5 Noted health effects not applicable for products with < 7% diuron, and applied to foliage.

6 Health hazards of specific pyrethroids vary, the effects indicated here represent those with most hazardous potential effects.

Appendix C

Online Resources & Tools

This compilation highlights a number of key online resources available through government agencies and public interest groups. It is not intended to be comprehensive.

Pesticide use data

California pesticide use reporting: *calpip.cdpr.ca.gov*

EPA Pesticide Industry Sales & Usage: *www.epa.gov/opp00001/pestsales*

USDA National Agricultural Statistics Service: *www.nass.usda.gov*

Pesticide health harms

Agency for Toxic Substances & Disease Registry, ToxFAQs: *www.atsdr.cdc.gov/az/c.html*

Collaborative on Health & the Environment, Toxicant & Disease Database: *www.healthandenvironment.org/tddb*

EPA Pesticides & Human Health Issues: *www.epa.gov/opp00001/health/human.htm*

EPA Recognition & Management of Pesticide Poisonings: *npic.orst.edu/rmpp.htm*

Ontario College of Family Physicians, Systematic Review of Pesticide Human Health Effects: *www.ocfp.on.ca/docs/pesticides-paper/pesticides-paper.pdf*

PAN International Highly Hazardous Pesticides: *www.panna.org/issues/publication/pan-international-list-highly-hazardous-pesticides*

PAN's pesticide database: *www.pesticideinfo.org*

Physicians for Social Responsibility, Pesticides & Human Health: A Resource For Health Care Professionals: *www.psr-la.org/resources/reports-training-materials/#Pesticides*

The Endocrine Disruption Exchange (TEDX): *www.endocrinedisruption.com/pesticides.introduction.php*

Pesticides & children's health

Beyond Pesticides, Learning/Developmental Disorders resource page: *www.beyondpesticides.org/health/learningdevelopmental.htm*

Center for Environmental Research & Children's Health: *cerch.org/research-programs/chamacos*

EPA Pesticides & Children: *www.epa.gov/opp00001/health/children.htm*

National Academy of Sciences: *www.nap.edu/catalog.php?record_id=2126*

PAN's Children's health page: *www.panna.org/children*

Pesticide food residues

FDA Total Diet Study: *www.fda.gov/Food/FoodSafety/FoodContaminantsAdulteration/TotalDietStudy/default.htm*

Whats On My Food? database (also includes health effect data): *www.whatsonmyfood.org*

USDA Pesticide Data Program: *www.ams.usda.gov/AMSv1.0/pdp*

Childhood disease & disorders

American Academy of Pediatrics: *www.aap.org*

CDC Child Health Statistics: *www.cdc.gov/nchs/fastats/children.htm*

Children's environmental health

Children's Environmental Health Network: *www.cehn.org*—A national multidisciplinary organization whose mission is to protect the developing child from environmental health hazards and promote a healthier environment.

Children's Environmental Health Project: *www.cape.ca/children*—A project of the Canadian Association of Physicians for the Environment, CEHP is intended to introduce clinicians (and their patients) to children's environmental health issues. Information on the health effects from environmental exposures is presented in a systems approach.

Healthy Child, Healthy World: *healthychild.org*—Protecting children's health and wellbeing from harmful environmental exposures through education and prevention strategies.

Healthy Kids: *www.healthy-kids.info*—*Provides resources and programs to help educators, health p*rofessionals, community officials, organizations, policy makers and parents work together to ensure schools are safe for children's healthy development.

Learning & Developmental Disabilities Initiative: *www.healthandenvironment.org/initiatives/learning*—An international partnership fostering collaboration among LDD organizations, researchers, health professionals and environmental health groups to address concerns about the impact environmental pollutants may have on children's neurological health.

Making our Milk Safe (MOMS): *www.safemilk.org*—A national grassroots movement of mothers working to create a healthier, safer environment for children, MOMS engages in education, advocacy and corporate campaigns.

Pediatric Environmental Health Specialty Units: *www.aoec.org/PEHSU.htm*—ATSDR and EPA support this network to provide education for health professionals, public health officials and others about the topic of children's environmental health.

Physicians for Social Responsibility: *www.psr.org/resources/pediatric-toolkit.html*—PSR has developed a pediatric environmental health toolkit that combines easy-to-use reference guides for health providers and user-friendly health education materials on preventing exposures to toxic chemicals and other substances that affect infant and child health. The toolkit is endorsed by the American Academy of Pediatrics.

Safer Chemicals, Healthy Families: *www.saferchemicals.org*—A coalition pressing for reform of national chemicals policy. SCHF represents more than 11 million individuals including parents, health professionals, advocates for people with learning and developmental disabilities, reproductive health advocates, environmentalists and businesses.

The Children's Environmental Health Institute: *cehi.org*—Works to identify, validate and develop solutions to address adverse health effects to children occurring as a consequence of exposure to hazardous environmental substances.

Pesticide Action Network North America
1611 Telegraph Ave, Suite 1200
Oakland CA 94612-2130
510.788.9020